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NEWS 8 Apr 22 Federal Research in Progress (FEDRIP) now available
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NEWS 14 Jul 29 Enhanced polymer searching in REGISTRY
NEWS 15 Jul 30 NETFIRST to be removed from STN
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NEWS 17 Aug 08 PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18 Aug 08 NTIS has been reloaded and enhanced
NEWS 19 Aug 19 Aquatic Toxicity Information Retrieval (AQUIRE)
now available on STN
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NEWS 22 Aug 26 Sequence searching in REGISTRY enhanced
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NEWS 25 Sep 16 CA Section Thesaurus available in CAPLUS and CA
NEWS 26 Oct 01 CASREACT Enriched with Reactions from 1907 to 1985
NEWS 27 Oct 21 EVENTLINE has been reloaded
NEWS 28 Oct 24 BEILSTEIN adds new search fields
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NEWS 30 Oct 25 MEDLINE SDI run of October 8, 2002
NEWS 31 Nov 18 DKILIT has been renamed APOLLIT
NEWS 32 Nov 25 More calculated properties added to REGISTRY
NEWS 33 Dec 02 TIBKAT will be removed from STN
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NEWS 36 Dec 17 TOXCENTER enhanced with additional content
NEWS 37 Dec 17 Adis Clinical Trials Insight now available on STN
NEWS 38 Dec 30 ISMEC no longer available
NEWS 39 Jan 13 Indexing added to some pre-1967 records in CA/CAPLUS
NEWS 40 Jan 21 NUTRACEUT offering one free connect hour in February 2003
NEWS 41 Jan 21 PHARMAML offering one free connect hour in February 2003
NEWS 42 Jan 29 Simultaneous left and right truncation added to COMPENDEX,
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NEWS EXPRESS January 6 CURRENT WINDOWS VERSION IS V6.01a,
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	ENTRY	SESSION
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FILE LAST UPDATED: 29 Jan 2003 (20030129/ED)

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=> s wo-0063189?/pn
L1 0 WO-0063189?/PN
(WO63189?/PN)

=> s wo-200063189?/pn
L2 2 WO-200063189?/PN
(WO200063189/PN)

=> p 1-2 pi

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L2 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000063191	A1	20001026	WO 1999-IB681	19990416
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9929536	A1	20001102	AU 1999-29536	19990416
WO 2000063189	A1	20001026	WO 2000-DK188	20000417 <--
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EP 1171430	A1	20020116	EP 2000-917222	20000417
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US 2002173647	A1	20021121	US 2002-142857	20020509
US 2003004341	A1	20030102	US 2002-209567	20020730

L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS

PATENT NO. KIND DATE APPLICATION NO. DATE

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PI  WO 2000063189      A1  20001026      WO 2000-DK188      20000417 <--
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WO 2000063191      A1  20001026      WO 1999-IB681      19990416
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      CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
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=> d 1-2

L2 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:756690 CAPLUS
 DN 133:310139
 TI Preparation of novel polymorphic forms of an antidiabetic agent
 IN Gaddam, Om Reddy; Potlapally, Rajender Kumar; Sirisilla, Raju;
 Krishnamurthi, Vyas; Dharmaraja, Sreenivasa Rao; Mamillapalli, Ramabhadra
 Sarma
 PA Reddy's Research Foundation, India
 SO PCT Int. Appl., 81 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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	US 2002173647	A1	20021121	US 2002-142857	20020509
	US 2003004341	A1	20030102	US 2002-209567	20020730
PRAI	WO 1999-IB681	A	19990416		
	IN 1999-MA436	A	19990419		
	DK 1999-536	A	19990420		

US 1999-132636P P 19990505
US 2000-550843 B1 20000417
US 2000-550862 A3 20000417
WO 2000-IB470 W 20000417

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
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L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
 AN 2000:756688 CAPLUS
 DN 133:310138
 TI Preparation of crystalline R-guanidines, arginine or L-arginine
 (2S)-2-ethoxy-3-[4-[2-(10H-phenoxazin-10-yl)ethoxy]phenyl]propanoate
 IN Ebdrup, Soren; Lugstein, Petra Christine
 PA Novo Nordisk A/S, Den.; Reddy's Research Foundation
 SO PCT Int. Appl., 34 pp.
 CODEN: PIXXD2
 DT Patent
 LA English
 FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000063189	A1	20001026	WO 2000-DK188	20000417 <--
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PRAI	WO 1999-IB681	W	19990416		
	DK 1999-536	A	19990420		
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☐ 1: Prostaglandins Leukot Essent Fatty Acids. 1999 May-Jun;60 (5-6):339-43.

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The role of the peroxisome proliferator-activated receptor alpha (PPAR alpha) in the control of cardiac lipid metabolism.

Djouadi F, Brandt JM, Weinheimer CJ, Leone TC, Gonzalez FJ, Kelly DP.

INSERM U319, Universite Paris 7, France.

The postnatal mammalian heart uses mitochondrial fatty acid oxidation (FAO) as the chief source of energy to meet the high energy demands necessary for pump function. Flux through the cardiac FAO pathway is tightly controlled in accordance with energy demands dictated by diverse physiologic and dietary conditions. In this report, we demonstrate that the lipid-activated nuclear receptor, peroxisome proliferator-activated receptor alpha (PPARalpha), regulates the expression of several key enzymes involved in cardiac mitochondrial FAO. In response to the metabolic stress imposed by pharmacologic inhibition of mitochondrial long-chain fatty acid import with etomoxir, PPARalpha serves as a molecular 'lipostat' factor by inducing the expression of target genes involved in fatty acid utilization including enzyme involved in mitochondrial and peroxisomal beta-oxidation pathways. In mice lacking PPARalpha (PPARalpha^{-/-} mice), etomoxir precipitates a cardiac phenotype characterized by myocyte lipid accumulation. Surprisingly, this metabolic regulatory response is influenced by gender as demonstrated by the observation that male PPARalpha^{-/-} mice are more susceptible to the metabolic stress compared to female animals. These results identify an important role for PPARalpha in the control of cardiac lipid metabolism.

PMID: 10471118 [PubMed - indexed for MEDLINE]



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Comment in:

- J Cardiovasc Risk. 2001 Aug;8(4):185-6.



PPARS, insulin resistance and type 2 diabetes.

Kaplan F, Al-Majali K, Betteridge DJ.Department of Medicine, Royal Free and University College Medical School
The Middlesex Hospital, London, UK.

It is clear that the PPAR receptors are exciting targets for therapeutic compounds likely to impact on insulin sensitivity, lipid and glucose homeostasis and vascular disease. The PPARgamma receptor agonists rosiglitazone and pioglitazone are very useful additions to the treatment options for type 2 diabetes. Currently they have limited licences, particularly in Europe, and hopefully as further clinical trial data becomes available these will be extended. Clinical outcome studies are important to ensure that the surrogate effects on glucose and other parameters translate into improved outcomes. There is exciting potential for these agents with the possibility of a combination of effects not only on glucose and lipid homeostasis but also on coagulation and thrombosis, blood pressure and microalbuminuria, which are likely to impact on vascular disease. If the current lack of evidence of serious hepatic toxicity persists they have an advantage over metformin in terms of tolerability and can be used in patients with impaired renal function. In addition to potential effects on diabetic outcome it will be of tremendous interest to determine whether these compounds, which improve insulin sensitivity and beta-cell function, will impact on the natural history of the disease. From what is known of the PPAR receptor systems it is likely that compounds acting as agonists or partial agonists for these receptors will have differing effects and it is possible to envisage the tailoring of compounds to enhance wanted effects and diminish unwanted effects, particularly fluid retention and weight gain. The future certainly looks exciting in this area.

Publication Types:

- Review
- Review, Tutorial

PMID: 11550999 [PubMed - indexed for MEDLINE]

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The human peroxisome proliferator-activated receptor alpha gene: identifica and functional characterization of two natural allelic variants.

Pharmacogenetics. 2000 Jun;10(4):321-33.

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New pharmacological agents under clinical investigation for treating disorders of lipoprotein regulation leading to atherosclerosis.

Gallup E, Dujovne C.

Radiant Research, Kansas Foundation for Clinical Pharmacology, 12200 W. 106th St., Ste. 330, Overland Park, KS 66215, USA.

Coronary heart disease (CHD), whose primary aetiology is atherosclerosis, is the leading cause of mortality and a major cause of morbidity in the industrialised world [1]. Serum lipoprotein levels are aetiologically related to the risk of atherosclerosis and CHD [2]. The liver and the gastrointestinal system are the major protagonists involved in regulation of lipoprotein biochemical-physiological mechanisms and the development of hypercholesterolaemia. Furthermore, specific lipoprotein receptors are being discovered as targets for pharmacological intervention to correct lipoprotein disorders. Agents that target lipoprotein regulation in the liver, gastrointestinal-biliary and atherosclerotic tissues resulting in improved serum lipoprotein levels and/or control of primary and secondary dyslipidaemic disorders including diabetes, are currently undergoing clinical trials. The most novel promising compounds, after the greatly effective newest HMG-CoA reductase inhibitors, are drugs that affect peroxisome proliferator-activated receptors, PPARalpha and PPARgamma receptors, bile acid transport mechanisms, cholesterol absorption and cholesterol acyltransferase and other biochemical targets of lipoprotein regulation. Current knowledge and ongoing trials with these agents are described here within the boundaries of investigator confidentiality agreements.

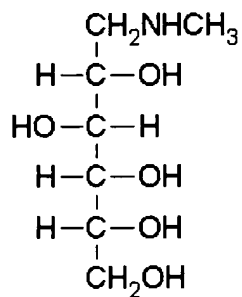
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